	Document ID	Title
1	US 20040115695 A1	Methods for generating enhanced antibody producing cell lines with improved growth characteristics
2	US 20040018550 A1	Antibodies immunologically specific for a DNA repair endonuclease and methods of use thereof
3	US 20030091997 A1	Chemical inhibitors of mismatch repair
4	US 20020123149 A1	Methods for generating hypermutable yeast
5	US 20020119439 A1	Genetic assays for trinucleotide repeat mutations in eukaryotic cells
6	US 20020068284 A1	Methods for generating hypermutable microbes
7	US 20020055106 A1	Method for generating hypermutable organisms
8	US 6599700 B1	Methods for detection of transition single-nucleotide polymorphisms
9	US 6576468 B1	Methods for obtaining microbe-resistant mammalian cells from hypermutable mammalian cells
10	US 6489115 B2	Genetic assays for trinucleotide repeat mutations in eukaryotic cells

	Document ID	Title
1	US 20050054056 A1	Variant cell surface molecule associated with cancer
2	US 20050048621 A1	Genetically altered antibody-producing cell lines with improved antibody characteristics
3	US 20040235108 A1	Monoclonal antibodies that specifically bind a tumor antigen
4	US 20040214288 A1	Methods of generating high-production of antibodies from hybridomas created by in vitro immunization
5	US 20040092021 A1	Methods for generating hypermutable yeast
6	US 20030176386 A1	Method for generating engineered cells for locus specific gene regulation and analysis
7 ✓	US 20030170895 A1	Methods of making hypermutable cells using PMSR homologs
8	US 20030143586 A1	Genetic hypermutability of plants for gene discovery and diagnosis
9	US 20030082511 A1	Identification of modulatory molecules using inducible promoters
10	US 20030068808 A1	Methods for generating antibiotic resistant microbes and novel antibiotics
11	US 20020123149 A1	Methods for generating hypermutable yeast

	Document ID	Title
12	US 20020068284 A1	Methods for generating hypermutable microbes
13	US 6921666 B2	Methods for generating hypermutable yeast
14	US 6656736 B2	Methods for generating hypermutable yeast

	Document ID	Title
1	US 20050054056 A1	Variant cell surface molecule associated with cancer
2	US 20050054048 A1	Antibodies and methods for generating genetically altered antibodies with enhanced effector function
3	US 20050048621 A1	Genetically altered antibody-producing cell lines with improved antibody characteristics
4	US 20040235108 A1	Monoclonal antibodies that specifically bind a tumor antigen
5	US 20040214288 A1	Methods of generating high-production of antibodies from hybridomas created by in vitro immunization
6	US 20030186441 A1	Methods for isolating novel antimicrobial agents from hypermutable mammalian cells
7	US 20030176386 A1	Method for generating engineered cells for locus specific gene regulation and analysis
8	US 20030170895 A1	Methods of making hypermutable cells using PMSR homologs
9	US 20030143586 A1	Genetic hypermutability of plants for gene discovery and diagnosis
10	US 20030068808 A1	Methods for generating antibiotic resistant microbes and novel antibiotics

	Document ID	Title
11	US 20020068284 A1	Methods for generating hypermutable microbes
12	US 20020055106 A1	Method for generating hypermutable organisms
13	US 6825038 B2	Method for generating hypermutable organisms
14	US 6576468 B1	Methods for obtaining microbe-resistant mammalian cells from hypermutable mammalian cells
15	US 6146894 A	Method for generating hypermutable organisms
16	WO 2005056599 A2	ANTIBODIES THAT SPECIFICALLY BIND PMS2

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	34835	(|435/471|435/6|435/252.1|.ccls.)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:17
L2	89	l1 and pms2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:17
L3	55	l2 and (bacteria or bacterium)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:17
L4	2	l3 and pmsr	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:18
L5	10	l3 and hypermutable	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:18
L6	7	l3 and nicolaides.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:18
L7	30	hypermutable same bacteri$2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L8	430	(mutH or muts or mutL or mutY or pms2 or mlh1 or mlh3 or msh2 or pmsr or pmsl) and "dominant negative"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L9	94	(mutH or muts or mutL or mutY or pms2 or mlh1 or mlh3 or msh2 or pmsr or pmsl) and hypermuta$4	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L10	0	(mutH or muts or mutL or mutY or pms2 or mlh1 or mlh3 or msh2 or pmsr or pmsl) and bacteri@2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L11	0	(mutH or muts or mutL or mutY or pms2 or mlh1 or mlh3 or msh2 or pmsr or pmsl) same bacteri@2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L12	37	(mutH or muts or mutL or mutY or pms2 or mlh1 or mlh3 or msh2 or pmsr or pmsl) same hypermuta$4	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19

L13	221	bacter?	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L14	238737	bacteria	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L15	239061	bacteri?	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L16	126	hypermutable	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L17	232	pms2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L18	33	pmsr or pms2l	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L19	43	hypermutable and (pms2 or (pmsr or pms2l))	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L20	30	(hypermutable and (pms2 or (pmsr or pms2l))) and bacteri?	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L21	2	"5846923".pn.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L22	16	nick.IN. and "regulatory elements"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L23	430	(mutH or muts or mutL or mutY or pms2 or mlh1 or mlh3 or msh2 or pmsr or pmsl) and "dominant negative"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L24	94	(mutH or muts or mutL or mutY or pms2 or mlh1 or mlh3 or msh2 or pmsr or pmsl) and hypermuta$4	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19

L25	0	(mutH or muts or mutL or mutY or pms2 or mlh1 or mlh3 or msh2 or pmsr or pmsl) and bacteri@2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L26	0	(mutH or muts or mutL or mutY or pms2 or mlh1 or mlh3 or msh2 or pmsr or pmsl) same bacteri@2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L27	37	(mutH or muts or mutL or mutY or pms2 or mlh1 or mlh3 or msh2 or pmsr or pmsl) same hypermuta$4	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L28	221	bacter?	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L29	238737	bacteria	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L30	239061	bacteri?	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L31	126	hypermutable	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L32	232	pms2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L33	30	hypermutable same bacteri$2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L34	33	pmsr or pms2l	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L35	43	hypermutable and (pms2 or (pmsr or pms2l))	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L36	30	(hypermutable and (pms2 or (pmsr or pms2l))) and bacteri?	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19

L37	2	"5846923".pn.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L38	16	nick.IN. and "regulatory elements"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L39	2681	mismatch near2 repair	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L40	232	pms2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L41	14	pmsr3	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L42	71	nicolaides.in. and sass.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L43	57	L42 and L31	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L44	35	L43 and pms2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19
L45	17	pms2-134	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/31 17:19

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     FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 17:22:30 ON 31 AUG 2005
L1         27611 S NICOLAIDES?/AU OR SASS?/AU OR GRASSO?/AU OR KINZLER?/AU OR VO
L2         11343 S "MISMATCH REPAIR"
L3            51 S (HYPERMUTABLE OR MUTAGENIZE OR MUTATED OR MUTATE) (2W) (BACTE
L4           698 S PMS2
L5            44 S PMSR
L6           739 S L4 OR L5
L7           600 S L6 AND L2
L8             0 S L7 AND L3
L9           740 S PMS2 OR PMSR OR PMS2L
L10      2481738 S PROKARYOTE OR BACTER?
L11         3861 S L2 (2W) GENE?
L12          398 S L AND L2
L13           17 S L12 AND L4
L14            7 S L13 NOT PY>=2002
L15            3 DUP REM L14 (4 DUPLICATES REMOVED)
L16            0 S L12 AND L5
L17            3 S L5 AND L2
L18            1 DUP REM L17 (2 DUPLICATES REMOVED)
L19         8938 S L10 AND (HYPERMUTABLE OR MUTAGENIZE OR MUTATED OR MUTATE)
L20          114 S L19 AND L2
L21            2 S L20 AND L4
L22            0 S L20 AND L5
L23            2 DUP REM L21 (0 DUPLICATES REMOVED)
```

L15 ANSWER 1 OF 3 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2001462119 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11506498
TITLE: Functional analysis of the **mismatch repair** system in bladder cancer.
AUTHOR: Thykjaer T; Christensen M; Clark A B; Hansen L R; Kunkel T A; Orntoft T F
CORPORATE SOURCE: Department of Clinical Biochemistry, Skejby University Hospital, 8200 Aarhus N, Denmark.
SOURCE: British journal of cancer, (2001 Aug 17) 85 (4) 568-75.
Journal code: 0370635. ISSN: 0007-0920.
PUB. COUNTRY: Scotland: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200109
ENTRY DATE: Entered STN: 20010820
Last Updated on STN: 20010924
Entered Medline: 20010920

AB In bladder cancer the observed microsatellite instability indicates that **mismatch repair** deficiency could be a frequently involved factor in bladder cancer progression. To investigate this hypothesis we analysed extracts of seven bladder cancer cell lines and, as a novel approach, five clinical cancer samples for **mismatch repair** activity. We found that one cell line (T24) and three of the clinical samples had a reduced repair capacity, measured to approximately 20% or less. The T24 cell extract was unable to repair a G-G mismatch and showed reduced repair of a 2-base loop, consistent with diminished function of the MSH2-MSH6 heterodimer. The functional assay was combined with measurement for mutation frequency, microsatellite analysis, sequencing, MTT assay, immunohistochemical analysis and RT-PCR analysis of the **mismatch repair** genes MSH2, MSH3, MSH6, PMS1, **PMS2** and MLH1. A >7-fold relative increase in mutation frequency was observed for T24 compared to a bladder cancer cell line with a fully functional **mismatch repair** system. Neither microsatellite instability, loss of repair nor **mismatch repair** gene mutations were detected. However, RT-PCR analysis of mRNA levels did detect changes in the ratio of expression of the Mut S and Mut **L** homologues. The T24 cell line had the lowest MSH6 expression level of the cell lines tested. Identical RT-PCR analysis of seventeen clinical samples (normal urothelium, 7; pTa low stage, 5; and pT1-4 high stage, 5) indicated a significant change in the expression ratio between MSH3/MSH6 (P< 0.004), MSH2/MSH3 (P< 0.012) and **PMS2** /MLH1 P< 0.005, in high stage bladder tumours compared to normal urothelium and low stage tumours. Collectively, the data suggest that imbalanced expression of **mismatch repair** genes could lead to partial loss of **mismatch repair** activity that is associated with invasive bladder cancer.

L15 ANSWER 2 OF 3 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 1999358298 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10429667
TITLE: Different **mismatch repair** deficiencies all have the same effects on somatic hypermutation: intact primary mechanism accompanied by secondary modifications.
AUTHOR: Kim N; Bozek G; Lo J C; Storb U
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, University of Chicago, Illinois 60637, USA.
CONTRACT NUMBER: GM07183 (NIGMS)
GM38649 (NIGMS)
SOURCE: Journal of experimental medicine, (1999 Jul 5) 190 (1) 21-30.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990910
Last Updated on STN: 19990910
Entered Medline: 19990824

AB Somatic hypermutation of Ig genes is probably dependent on transcription of the target gene via a mutator factor associated with the RNA polymerase (Storb, U., E.L. Klotz, J. Hackett, Jr., K. Kage, G. Bozek, and T.E. Martin. 1998. J. Exp. Med. 188:689-698). It is also probable that some form of DNA repair is involved in the mutation process. It was shown that the nucleotide excision repair proteins were not required, nor were **mismatch repair** (MMR) proteins. However, certain changes in mutation patterns and frequency of point mutations were observed in Msh2 (MutS homologue) and **Pms2** (MutL homologue) MMR-deficient mice (for review see Kim, N., and U. Storb. 1998. J. Exp. Med. 187:1729-1733). These data were obtained from endogenous immunoglobulin (Ig) genes and were presumably influenced by selection of B cells whose Ig genes had undergone certain mutations. In this study, we have analyzed somatic hypermutation in two MutL types of MMR deficiencies, **Pms2** and Mlh1. The mutation target was a nonselectable Ig-kappa gene with an artificial insert in the V region. We found that both **Pms2**- and Mlh1-deficient mice can somatically hypermutate the Ig test gene at approximately twofold reduced frequencies. Furthermore, highly mutated sequences are almost absent. Together with the finding of genome instability in the germinal center B cells, these observations support the conclusion, previously reached for Msh2 mice, that MMR-deficient B cells undergoing somatic hypermutation have a short life span. **Pms2**- and Mlh-1-deficient mice also resemble Msh2-deficient mice with respect to preferential targeting of G and C nucleotides. Thus, it appears that the different MMR proteins do not have unique functions with respect to somatic hypermutation. Several intrinsic characteristics of somatic hypermutation remain unaltered in the MMR-deficient mice: a preference for targeting A over T, a strand bias, mutational hot spots, and hypermutability of the artificial insert are all seen in the unselectable Ig gene. This implies that the MMR proteins are not required for and most likely are not involved in the primary step of introducing the mutations. Instead, they are recruited to repair certain somatic point mutations, presumably soon after these are created.

L15 ANSWER 3 OF 3 MEDLINE on STN
ACCESSION NUMBER: 97463808 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9322509
TITLE: Mutations predisposing to hereditary nonpolyposis colorectal cancer: database and results of a collaborative study. The International Collaborative Group on Hereditary Nonpolyposis Colorectal Cancer.
AUTHOR: Peltomaki P; Vasen H F
CORPORATE SOURCE: Department of Medical Genetics, Haartman Institute, Helsinki, Finland.. Paivi.Peltomaki@helsinki.fi
CONTRACT NUMBER: CA67941 (NCI)
SOURCE: Gastroenterology, (1997 Oct) 113 (4) 1146-58. Journal code: 0374630. ISSN: 0016-5085.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (MULTICENTER STUDY)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 19971105
Last Updated on STN: 20020420
Entered Medline: 19971023

AB BACKGROUND & AIMS: Germline mutations in four DNA **mismatch repair** genes are known to cause susceptibility to hereditary nonpolyposis colorectal cancer (HNPCC). The rapidly increasing information about these mutations needs to be collected and appropriately stored to facilitate further studies on the biological and clinical significance of the findings. METHODS: The International Collaborative Group on HNPCC has established a database of DNA **mismatch**

repair gene mutations and polymorphisms. In this report, 126 predisposing mutations were analyzed. RESULTS: A majority of the mutations affected either the Mut **L** homologue (MLH) 1 (n = 75) or the Mut S homologue (MSH) 2 (n = 48) and were quite evenly distributed, with some clustering in MSH2 exon 12 and MLH1 exon 16. Most MSH2 mutations consisted of frameshift (60%) or nonsense changes (23%), whereas MLH1 was mainly affected by frameshift (40%) or missense alterations (31%). Although most mutations were unique, a few common recurring mutations were identified. Of the families studied (n = 202), 82% met the Amsterdam criteria and 15% did not; the general mutation profile was similar in both groups. CONCLUSIONS: The construction of mutation profiles will facilitate the development of diagnostic strategies in HNPCC.

=>

L18 ANSWER 1 OF 1 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 1998008931 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9344666
TITLE: PMS2-related genes flank the rearrangement breakpoints associated with Williams syndrome and other diseases on human chromosome 7.
AUTHOR: Osborne L R; Herbrick J A; Greavette T; Heng H H; Tsui L C; Scherer S W
CORPORATE SOURCE: Department of Genetics, The Hospital for Sick Children, Toronto, Ontario M5G 1X8, Canada.
SOURCE: Genomics, (1997 Oct 15) 45 (2) 402-6.
Journal code: 8800135. ISSN: 0888-7543.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980224
Last Updated on STN: 20020420
Entered Medline: 19980212

AB The human PMS2 **mismatch repair** gene and a family of at least 17 other related genes (named human **PMSR** or PMS2L genes) have been localized to human chromosome 7. Human PMS2 has been mapped previously to 7p22 and shown to be causative in hereditary nonpolyposis colon cancer (HNPCC), but the human PMS2L genes have not been positioned in the context of the physical or genetic map of chromosome 7. In this study we have used various mapping methodologies to determine the precise location of the human PMS2L genes at 7q11.22, 7q11.23, and 7q22. Within 7q11.23, human PMS2L genes were found to be present at at least three sites as part of duplicated genomic segments that flank the most common rearrangement breakpoints in Williams syndrome.

=>

L23 ANSWER 1 OF 2 MEDLINE on STN
ACCESSION NUMBER: 2001431932 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11479923
TITLE: Loss of **mismatch repair** activity in simian virus 40 large T antigen-immortalized BPH-1 human prostatic epithelial cell line.
AUTHOR: Yeh C C; Lee C; Huang M C; Dahiya R
CORPORATE SOURCE: Department of Urology, Veterans Affairs Medical Center and University of California, San Francisco, California, USA.
CONTRACT NUMBER: AG16870 (NIA)
CA64872 (NCI)
DK47517 (NIDDK)
SOURCE: Molecular carcinogenesis, (2001 Jul) 31 (3) 145-51.
Journal code: 8811105. ISSN: 0899-1987.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010903
Last Updated on STN: 20030225
Entered Medline: 20010830

AB Simian virus 40 large T antigen (SVLTAg) has been used to immortalize cells; however, the mechanism leading to immortalization is still unclear. We hypothesize that DNA **mismatch repair** (MMR) activity is important during SVLTAg-induced immortalization. To test this hypothesis, we used the SVLTAg-immortalized cell line BPH-1 derived from human benign prostate epithelial cells to analyze MMR activity and the expression of MMR genes (hMLH1, hPMS1, hPMS2, hMSH2, hMSH3, and hMSH6). The results demonstrated that BPH-1 cells were deficient in repairing G:T, A:C, and G:G mispairs in **bacteriophage** M13mp2. Reverse-transcription polymerase chain reaction experiments indicated MMR genes (hMSH3, hMSH6, and hPMS1) were expressed at a low level in BPH-1 cells. In contrast, all six MMR genes were expressed in human benign prostate hyperplasia tissues. Downregulation of hMSH3, hMSH6, and hPMS1 genes is not a result of the hypermethylation mechanism because demethylation with 5-aza-2'-deoxycytidine did not restore expression of these genes. Although the hMLH1 gene is expressed in BPH-1 cells, western blotting and exon analyses demonstrated that hMLH1 was **mutated** and/or deleted in BPH-1 cells.

L23 ANSWER 2 OF 2 MEDLINE on STN
ACCESSION NUMBER: 94352394 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8072530
TITLE: Mutations of two PMS homologues in hereditary nonpolyposis colon cancer.
AUTHOR: Nicolaides N C; Papadopoulos N; Liu B; Wei Y F; Carter K C; Ruben S M; Rosen C A; Haseltine W A; Fleischmann R D; Fraser C M; +
CORPORATE SOURCE: Johns Hopkins Oncology Center, Baltimore, Maryland 21231.
SOURCE: Nature, (1994 Sep 1) 371 (6492) 75-80.
Journal code: 0410462. ISSN: 0028-0836.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U13695; GENBANK-U13696
ENTRY MONTH: 199409
ENTRY DATE: Entered STN: 19941006
Last Updated on STN: 20020420
Entered Medline: 19940923

AB Hereditary nonpolyposis colorectal cancer (HNPCC) is one of man's commonest hereditary diseases. Several studies have implicated a defect in DNA **mismatch repair** in the pathogenesis of this disease. In particular, hMSH2 and hMLH1 homologues of the **bacterial** DNA **mismatch repair** genes mutS and

mutL, respectively, were shown to be **mutated** in a subset of HNPCC cases. Here we report the nucleotide sequence, chromosome localization and mutational analysis of hPMS1 and hPMS2, two additional homologues of the prokaryotic mutL gene. Both hPMS1 and hPMS2 were found to be **mutated** in the germline of HNPCC patients. This doubles the number of genes implicated in HNPCC and may help explain the relatively high incidence of this disease.

=>

L29 ANSWER 1 OF 13 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2001690374 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11717398
TITLE: Mutation frequency and biological cost of **antibiotic resistance** in Helicobacter pylori.
AUTHOR: Bjorkholm B; Sjolund M; Falk P G; Berg O G; Engstrand L; Andersson D I
CORPORATE SOURCE: Swedish Institute for Infectious Disease Control, 17182 Solna, Sweden.
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (2001 Dec 4) 98 (25) 14607-12. Electronic Publication: 2001-11-20. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20011213
Last Updated on STN: 20030105
Entered Medline: 20020110

AB Among the several factors that affect the appearance and spread of acquired **antibiotic resistance**, the mutation frequency and the biological cost of resistance are of special importance. Measurements of the mutation frequency to rifampicin resistance in Helicobacter pylori strains isolated from dyspeptic patients showed that approximately 1/4 of the isolates had higher mutation frequencies than Enterobacteriaceae **mismatch-repair** defective mutants. This high mutation frequency could explain why resistance is so frequently acquired during antibiotic treatment of H. pylori infections. Inactivation of the mutS gene had no substantial effect on the mutation frequency, suggesting that MutS-dependent **mismatch repair** is absent in this **bacterium**. Furthermore, clarithromycin resistance conferred a biological cost, as measured by a decreased competitive ability of the resistant mutants in mice. In clinical isolates this cost could be reduced, indicating that compensation is a clinically relevant phenomenon that could act to stabilize resistant **bacteria** in a population.

L29 ANSWER 2 OF 13 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:567586 BIOSIS
DOCUMENT NUMBER: PREV200100567586
TITLE: An rpsL cassette, janus, for gene replacement through negative selection in Streptococcus pneumoniae.
AUTHOR(S): Sung, C. K.; Li, H.; Claverys, J. P.; Morrison, D. A. [Reprint author]
CORPORATE SOURCE: University of Illinois at Chicago, 900 South Ashland Ave., Room 4110, Chicago, IL, 60607, USA
DAMorris@uic.edu
SOURCE: Applied and Environmental Microbiology, (November, 2001) Vol. 67, No. 11, pp. 5190-5196. print. CODEN: AEMIDF. ISSN: 0099-2240.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Dec 2001
Last Updated on STN: 25 Feb 2002

AB Natural genetic transformation offers a direct route by which synthetic gene constructs can be placed into the single circular chromosome of Streptococcus pneumoniae. However, the lack of a general negative-selection marker has hampered the introduction of constructs that do not confer a selectable phenotype. A 1.3-kb cassette was constructed comprising a kanamycin (Kn) resistance marker (kan) and a counterselectable rpsL+ marker. The cassette conferred dominant streptomycin (Sm) sensitivity in an Sm-resistant background in S. pneumoniae. It was demonstrated that it could be used in a two-step transformation procedure to place DNA of arbitrary sequence at a chosen

target site. The first transformation into an Sm-resistant strain used the cassette to tag a target gene on the chromosome by homologous recombination while conferring Kn resistance but Sm sensitivity on the recombinant. Replacement of the cassette by an arbitrary segment of DNA during a second transformation restored Sm resistance (and Kn sensitivity), allowing construction of silent mutations and deletions or other gene replacements which lack a selectable phenotype. It was also shown that gene conversion occurred between the two rpsL alleles in a process that depended on recA and that was susceptible to correction by **mismatch repair**.

L29 ANSWER 3 OF 13 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2001294692 EMBASE
TITLE: **Mismatch repair** and virulence.
AUTHOR: Matic I.
CORPORATE SOURCE: matic@necker.fr
SOURCE: Trends in Microbiology, (1 Aug 2001) Vol. 9, No. 8, pp. 360.
Refs: 2
ISSN: 0966-842X CODEN: TRMIEA
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Note
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
ENTRY DATE: Entered STN: 20010906
Last Updated on STN: 20010906
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L29 ANSWER 4 OF 13 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:385094 BIOSIS
DOCUMENT NUMBER: PREV200100385094
TITLE: Mitochondria isolated from liver contain the essential factors required for RNA/DNA oligonucleotide-targeted gene repair.
AUTHOR(S): Chen, Zongyu; Felsheim, Rod; Wong, Phillip; Augustin, Lance B.; Metz, Richard; Kren, Betsy T.; Steer, Clifford J. [Reprint author]
CORPORATE SOURCE: Department of Medicine, 420 Delaware Street S.E., Mayo Building, Room A536, Minneapolis, MN, 55455, USA
steer001@tc.umn.edu
SOURCE: Biochemical and Biophysical Research Communications, (July 13, 2001) Vol. 285, No. 2, pp. 188-194. print.
CODEN: BBRCA9. ISSN: 0006-291X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Aug 2001
Last Updated on STN: 19 Feb 2002

AB Chimeric RNA/DNA oligonucleotides (ONs) have been used successfully for site-specific modifications of episomal and chromosomal DNA in eukaryotic cells. We explored the possibility of applying this technique to mitochondrial DNA, as single-nucleotide defects in this genome are associated with a series of human diseases. Therefore, we determined whether mitochondria possess the enzymatic machinery for chimeric ON-mediated DNA alterations. We utilized an in vitro DNA repair assay and an Escherichia coli read-out system with mutagenized plasmids carrying point mutations in **antibiotic resistance** genes.
RNA/DNA ONs were designed to correct the defects and restore kanamycin and tetracyclin resistance. Using this system, we demonstrated that extracts from highly purified rat liver mitochondria possess the essential enzymatic activity to mediate precise single-nucleotide changes. Interestingly, the frequency of gene conversion was similar in both mitochondrial and nuclear extracts, as well as from quiescent and regenerating liver. The results indicate that mitochondria contain the machinery required for repair of genomic single-point mutations, and suggest that RNA/DNA ONs may provide a novel approach to the treatment of certain mitochondrial-based diseases.

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on STN DUPLICATE 2

ACCESSION NUMBER: 2000281559 EMBASE
TITLE: Molecular biology of Streptococcus pneumoniae: An everlasting challenge.
AUTHOR: Sicard M.; Gasc A.M.; Giammarinaro P.; Lefrancois J.; Pasta F.; Samrakandi M.
CORPORATE SOURCE: M. Sicard, Lab. Microbiol. Gen. Molec. C.N.R.S., Universite Paul Sabatier, 118, route de Narbonne, 31062 Toulouse cedex, France. sicard@ibcg.biotoul.fr
SOURCE: Research in Microbiology, (2000) Vol. 151, No. 6, pp. 407-411.
Refs: 32
ISSN: 0923-2508 CODEN: RMCREW
COUNTRY: France
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20000831
Last Updated on STN: 20000831

AB Streptococcus pneumoniae is a model for elucidating: 1) recombination steps of DNA, from its discovery to polarity of integration; 2) long-patch **mismatch repair**, short-patch repair triggered by A/G and exclusion of deletions; 3) resistance to β-lactam antibiotics; and 4) factors of virulence. Several of these topics remain a challenge for future investigations. (C) 2000 Editions scientifiques et medicales Elsevier SAS.

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on STN

ACCESSION NUMBER: 1999418963 EMBASE
TITLE: Relative stabilities of dinucleotide and tetranucleotide repeats in cultured mammalian cells.
AUTHOR: Lee J.S.; Hanford M.G.; Genova J.L.; Farber R.A.
CORPORATE SOURCE: R.A. Farber, Dept. Pathology Laboratory Medicine, Univ. North Carolina at Chapel Hill, CB 7525, Chapel Hill, NC 27599-7525, United States. rfarber@med.unc.edu
SOURCE: Human Molecular Genetics, (1999) Vol. 8, No. 13, pp. 2567-2572.
Refs: 34
ISSN: 0964-6906 CODEN: HMGEE5
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 022 Human Genetics
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 19991216
Last Updated on STN: 19991216

AB The differences in rates of frameshift mutations between a dinucleotide repeat sequence [(CA)17] and a tetranucleotide repeat sequence [(GAAA)17] have been determined in immortalized, non-tumorigenic, **mismatch repair**-proficient mouse cells and in **mismatch repair**-defective human colorectal cancer cells. Clones with mutations were selected on the basis of restoration of activity of a **bacterial** neomycin resistance gene whose reading frame was disrupted by insertion of the microsatellite upstream of the translation initiation codon. This gene was introduced into the cells on a plasmid, which integrated into the genome of the host cells. Mutation rates of the tetranucleotide repeat were much lower than those of the dinucleotide repeat in both cell types. In addition, independent subclones of the colorectal cancer cell line were assayed by PCR for instability of endogenous tetranucleotide and dinucleotide repeat sequences. In all cases, the mutation frequencies of the dinucleotide repeats were higher than those of the tetranucleotide repeats.

L29 ANSWER 7 OF 13 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2000050653 MEDLINE

DOCUMENT NUMBER: PubMed ID: 10581263
TITLE: DNA sequence similarity requirements for interspecific recombination in Bacillus.
AUTHOR: Majewski J; Cohan F M
CORPORATE SOURCE: Department of Biology, Wesleyan University, Middletown, Connecticut 06459, USA.. majewski@complex.rockefeller.edu
SOURCE: Genetics, (1999 Dec) 153 (4) 1525-33.
Journal code: 0374636. ISSN: 0016-6731.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF172323
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000218
Last Updated on STN: 20000218
Entered Medline: 20000208

AB Gene transfer in **bacteria** is notoriously promiscuous. Genetic material is known to be transferred between groups as distantly related as the Gram positives and Gram negatives. However, the frequency of homologous recombination decreases sharply with the level of relatedness between the donor and recipient. Several studies show that this sexual isolation is an exponential function of DNA sequence divergence between recombining substrates. The two major factors implicated in producing the recombinational barrier are the **mismatch repair** system and the requirement for a short region of sequence identity to initiate strand exchange. Here we demonstrate that sexual isolation in Bacillus transformation results almost exclusively from the need for regions of identity at both the 5' and 3' ends of the donor DNA strand. We show that, by providing the essential identity, we can effectively eliminate sexual isolation between highly divergent sequences. We also present evidence that the potential of a donor sequence to act as a recombinogenic, invasive end is determined by the stability (melting point) of the donor-recipient complex. These results explain the exponential relationship between sexual isolation and sequence divergence observed in **bacteria**. They also suggest a model for rapid spread of novel adaptations, such as **antibiotic resistance** genes, among related species.

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ACCESSION NUMBER: 1999:236703 BIOSIS
DOCUMENT NUMBER: PREV199900236703
TITLE: Patterns of **antibiotic resistance** among **mismatch repair** deficient **bacterial** mutators.
AUTHOR(S): Levy, D. D. [Reprint author]; Cebula, T. A. [Reprint author]
CORPORATE SOURCE: Food and Drug Administration, 200 C St., SW Washington, DC, 20204, USA
SOURCE: Environmental and Molecular Mutagenesis, (1999) Vol. 33, No. SUPPL. 33, pp. 40. print.
Meeting Info.: 1999 Environmental Mutagen Society Meeting. Washington, D.C., USA. March 27-April 1, 1999. Environmental Mutagen Society.
CODEN: EMMUEG. ISSN: 0893-6692.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Jun 1999
Last Updated on STN: 17 Jun 1999

L29 ANSWER 9 OF 13 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:498533 BIOSIS
DOCUMENT NUMBER: PREV199800498533
TITLE: Induction of microsatellite instability by oxidative DNA damage.
AUTHOR(S): Jackson, Aimee L.; Chen, Ru; Loeb, Lawrence A. [Reprint author]

CORPORATE SOURCE: Dep. Pathol., Univ. Washington, Box 357705, Seattle, WA 98195, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (Oct. 13, 1998) Vol. 95, No. 21, pp. 12468-12473. print.
CODEN: PNASA6. ISSN: 0027-8424.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 18 Nov 1998
Last Updated on STN: 18 Nov 1998

AB Instability of repetitive sequences, both in intronic sequences and within coding regions, has been demonstrated to be a hallmark of genomic instability in human cancer. Understanding how these mutational events arise may provide an opportunity for prevention or early intervention in cancer development. To study the source of this instability, we have identified a region of the beta-lactamase gene that is tolerant to the insertion of fragments of exogenous DNA as large as 1,614 bp with minimal loss of enzyme activity, as determined by **antibiotic resistance**. Fragments inserted out-of-frame render Escherichia coli sensitive to antibiotic, and compensatory frameshift mutations that restore the reading frame of beta-lactamase can be selected on the basis of **antibiotic resistance**. We have utilized this site to insert a synthetic microsatellite sequence within the beta-lactamase gene and selected for mutations yielding frameshifts. This assay provides for detection of one frameshift mutation in a background of 106 wild-type sequences. **Mismatch repair** deficiency increased the observed frameshift frequency apprxeq300-fold. Exposure of plasmid containing microsatellite sequences to hydrogen peroxide resulted in frameshift mutations that were localized exclusively to the microsatellite sequences, whereas DNA damage by UV or N-methyl-N'-nitro-N-nitrosoguanidine did not result in enhanced mutagenesis. We postulate that in tumor cells, endogenous production of oxygen free radicals may be a major factor in promoting instability of microsatellite sequences. This beta-lactamase assay may provide a sensitive methodology for the detection and quantitation of mutations associated with the development of cancer.

L29 ANSWER 10 OF 13 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 97053611 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8895473
TITLE: High mutation frequencies among Escherichia coli and Salmonella pathogens.
COMMENT: Comment in: Science. 1996 Nov 15;274(5290):1081. PubMed ID: 8966583
Comment in: Science. 1997 Sep 19;277(5333):1833-4. PubMed ID: 9324769
AUTHOR: LeClerc J E; Li B; Payne W L; Cebula T A
CORPORATE SOURCE: Molecular Biology Branch, Center for Food Safety and Applied Nutrition (HFS-235), Food and Drug Administration, Washington, DC 20204, USA.. tac@vax8.cfsan.fda.gov
SOURCE: Science, (1996 Nov 15) 274 (5290) 1208-11.
Journal code: 0404511. ISSN: 0036-8075.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U29579; GENBANK-U69873
ENTRY MONTH: 199612
ENTRY DATE: Entered STN: 19970128
Last Updated on STN: 19990129
Entered Medline: 19961210

AB Here it is reported that the incidence of mutators among isolates of pathogenic Escherichia coli and Salmonella enterica is high (over 1 percent). These findings counter the theory, founded on studies with laboratory-attenuated strains, that suggests mutators are rare among **bacterial** populations. Defects in methyl-directed **mismatch repair** underlie all mutator phenotypes described here. Of nine independently derived hypermutable strains, seven contained a defective mutS allele. Because these mutant alleles increase

the mutation rate and enhance recombination among diverse species, these studies may help explain both the rapid emergence of **antibiotic resistance** and the penetrance of virulence genes within the prokaryotic community.

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ACCESSION NUMBER: 96235032 EMBASE
DOCUMENT NUMBER: 1996235032
TITLE: Homologous recombination between the tuf genes of Salmonella typhimurium.
AUTHOR: Abdulkarim F.; Hughes D.
CORPORATE SOURCE: Department of Molecular Biology, The Biomedical Center, Uppsala University,S-751 24 Uppsala, Sweden
SOURCE: Journal of Molecular Biology, (1996) Vol. 260, No. 4, pp. 506-522.
ISSN: 0022-2836 CODEN: JMOBAK
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 021 Developmental Biology and Teratology
022 Human Genetics
029 Clinical Biochemistry
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 961015
Last Updated on STN: 961015

AB The genes coding for the translation factor EF-Tu, tufA and tufB are separated by over 700 kb on the circular chromosome of Salmonella typhimurium. The coding regions of these genes have 99% identity at the nucleotide level in spite of the presumed ancient origin of the gene duplication. Sequence comparisons between S. typhimurium and Escherichia coli suggest that within each species the two tuf genes are evolving in concert. Here we show that each of the S. typhimurium tuf genes can transfer genetic information to the other. In our genetic system the transfers are seen as non-reciprocal, i.e. as gene conversion events. However, the mechanism of recombination could be reciprocal, with sister chromosome segregation and selection leading to the isolation of a particular class of recombinant. The amount of sequence information transferred in individual recombination events varies, but can be close to the entire length of the gene. The recombination is RecABCD-dependent, and is opposed by MutSHLU **mismatch repair**. In the wild-type, this type of recombination occurs at a rate that is two or three orders of magnitude greater than the nucleotide substitution rate. The rate of recombination differs by six orders of magnitude between a recA and a mutS strain. **Mismatch repair** reduces the rate of this recombination 1000-fold. The rate of recombination also differs by one order of magnitude depending on which tuf gene is donating the sequence selected for. We discuss three classes of model that could, in principle, account for the sequence transfers: (1) tuf mRNA mediated recombination; (2) non-allelic reciprocal recombination involving sister chromosomes; (3) non-allelic gene conversion involving sister chromosomes, initiated by a double-strand break close to one tuf gene. Although the mechanism remains to be determined, the effect on the **bacterial** cells is tuf gene sequence homogenisation. This recombination phenomenon can account for the concerted evolution of the tuf genes.

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ACCESSION NUMBER: 1984:64861 BIOSIS
DOCUMENT NUMBER: PREV198426064861; BR26:64861
TITLE: MOLECULAR CHARACTERIZATION OF GENE E INVOLVED IN **MISMATCH REPAIR** IN STREPTOCOCCUS-PNEUMONIAE.
AUTHOR(S): CLAVERYS J P [Reprint author]; GHERARDI M
CORPORATE SOURCE: CRBGC-CNRS 31062 TOULOUSE CEDEX, FR
SOURCE: Journal of Cellular Biochemistry Supplement, (1983) No. 7 PART B, pp. 220.

Meeting Info.: 12TH ANNUAL UCLA (UNIVERSITY OF CALIFORNIA-LOS ANGELES) SYMPOSIUM ON CELLULAR RESPONSES TO DNA DAMAGE, APR. 10-15, 1983. J CELL BIOCHEM.
ISSN: 0733-1959.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH

L29 ANSWER 13 OF 13 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1981:196334 BIOSIS
DOCUMENT NUMBER: PREV198171066326; BA71:66326
TITLE: BROMO URACIL INDUCED MUTAGENESIS IN A **MISMATCH REPAIR** DEFICIENT STRAIN OF HAEMOPHILUS-INFLUENZAE.
AUTHOR(S): BAGCI H [Reprint author]; STUY J H
CORPORATE SOURCE: DEP BIOL SCI, FLA STATE UNIV, TALLAHASSEE, FLA 32306, USA
SOURCE: Mutation Research, (1980) Vol. 73, No. 1, pp. 15-20.
CODEN: MUREAV. ISSN: 0027-5107.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB Cells of wild-type H. influenzae and of a **mismatch-repair**-deficient mutant (hex-) were grown in a chemically defined medium containing thymidine or 5-bromodeoxyuridine (BUdR). Spontaneous mutation frequencies to resistance against 3 antibiotics observed for the thymidine cultures were 10-30 times higher for the hex- mutant. The mutation frequencies observed for the BUdR hex- culture were increased by another 10 times while those for the wild-type suspension did not differ from the frequencies seen in the thymidine medium.

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